

U.S. Department of Labor

Office of Administrative Law Judges
Seven Parkway Center - Room 290
Pittsburgh, PA 15220

(412) 644-5754
(412) 644-5005 (FAX)



Issue date: 17Jul2001

CASE NO.: 2000-BLA-127

In the Matter of

ELSIE KOZELE, Surviving Spouse of
JOSEPH KOZELE
Claimant

v.

KEYSTONE COAL MINING CORP.
Employer

and

DIRECTOR, OFFICE OF WORKERS'
COMPENSATION PROGRAMS
Party-In-Interest

Appearances:

Heath Long, Esquire
For the Claimant

Hilary Daninhirsch, Esquire
For the Employer

Before: MICHAEL P. LESNIAK
Administrative Law Judge

DECISION AND ORDER – AWARDING BENEFITS

This case arises from a claim for benefits under the “Black Lung Benefits Act,” Title IV of the Federal Coal Mine Health and Safety Act of 1969, as amended, 30 U.S.C. § 901 *et seq.* (hereinafter referred to as “the Act”), and applicable federal regulations, mainly 20 C.F.R. Parts 410, 718 and 727 (“Regulations”).

Benefits under the Act are awarded to persons who are totally disabled within the meaning of the Act due to pneumoconiosis or to the survivors of persons whose death was caused by pneumoconiosis. Pneumoconiosis is a dust disease of the lung arising from coal mine employment and is commonly known as black lung.

A formal hearing was conducted in Pittsburgh, Pennsylvania on May 23, 2000, at which all parties were afforded a full opportunity to present evidence and argument, as provided in the Act and Regulations issued thereunder, found in Title 20, Code of Federal Regulations.¹

ISSUES

The sole issue is:

Whether the miner's death was due to pneumoconiosis.² (TR 10).

FINDINGS OF FACT AND CONCLUSIONS OF LAW

Procedural History and Factual Background

The miner, Joseph Kozele, filed his first claim for black lung benefits on February 2, 1979. (DX-25). On May 12, 1980, the miner was awarded state workers' compensation benefits for a partial disability due to coal worker's pneumoconiosis ("CWP"). On July 23, 1979, the Initial Notice of Finding indicated that the miner was entitled to benefits. On October 6, 1980, Administrative Law Judge ("ALJ") Vivian Schreter Murray issued a Decision and Order Denying Benefits. The miner subsequently appealed the decision to the Benefits Review Board ("Board"). In a published opinion, on June 10, 1983, the Board affirmed the Decision and Order of ALJ Murray. (DX-25).

The miner filed his second claim for benefits on December 12, 1996. (DX-26). On March 18, 1996, the miner was denied benefits by the claims examiner. The miner rejected the findings of the claims examiner and requested a formal hearing. By letter of November 5, 1997, the employer advised ALJ George P. Morin that they were conceding liability and that the case should be remanded for payment of benefits. (DX-26).

¹ At the hearing Director's exhibits 1-28, Claimant's exhibits 1- 9 and Employer's exhibits 1-6 were admitted into evidence without objection. (TR 5-7).

² The following abbreviations have been used in this opinion: DX = Director's exhibit, EX = Employer's exhibit, CX = Claimant's exhibit, TR = Transcript of the hearing, BCR = Board-certified radiologist, BCI = Board-certified internist, and B = B reader.

The miner died on July 31, 1998. (DX-3). Claimant filed the instant claim for survivor benefits on March 10, 1999. (DX-1).

At the hearing Claimant, Elsie Kozele testified. She testified that she married the miner in 1979. (TR 11). She has not remarried. (TR 11). Claimant noted that the miner left his coal mine employment in 1979. They met in 1976 or 1977. The miner was an active coal miner at that time. (TR 12). Before they got married the miner had trouble breathing and his breathing got worse over time. (TR 12-13). The miner had to give up hunting and fishing about six (6) months before he died. (TR 13). Claimant noticed that the miner was very short of breath about 5 years before he died. (TR 14). The miner was treated by Dr. Hanna for his breathing problem. (TR 14). While they were married, the miner was never a smoker. (TR 15). The miner had heart problems for which he had angioplasty and he had a bout of pneumonia. (TR 16). The miner also had a cough with sputum production that developed after they were married. (TR 17).

On cross-examination, the claimant acknowledged the miner was 83 when he died. (TR 18). She did not know whether the miner smoked prior to 1976. (TR 19). She noted that after a bout of pneumonia the miner was on oxygen for a short time. (TR 20). Claimant testified that the miner was treated for high blood pressure, but denied the miner had diabetes, prostate cancer, or a stroke. (TR 20). Claimant added that their marriage was the second for the miner, his first wife had died. (TR 22).

Medical Evidence

Death Certificate

The miner's death certificate was certified by Dr. Brent Ednie. (DX-3). The miner died on July 31, 1998. The cause of death was listed as (a) pneumonia and (b) pulmonary hypertension.

Autopsy Report

The autopsy was performed on August 1, 1998 by Drs. Pisano and Shenouda. (DX-4). Dr. James J. Pisano is Board-Certified in Anatomical and Clinical Pathology. (see curriculum vitae at DX-4). Final anatomical diagnoses included: severe three blood vessels coronary artery disease, early myocardial ischemia, old myocardial infarction, cardiomegaly, cor pulmonale, squamous cell carcinoma of the lungs, emphysema, pulmonary hypertension, diffuse interstitial fibrosis, macronodular coal workers' pneumoconiosis, and bilateral pleural effusion.

Gross examination of the lungs showed that the right lung weighed 950 grams and the left lung weighed 500 grams. The prosectors also noted multiple black firm nodules scattered in the pulmonary parenchyma, the largest measured 1 cm. in greatest dimension which were occupying approximately fifty percent (50%) of the pulmonary parenchyma.

Microscopic examination of lung tissue showed diffuse emphysema with diffuse areas showing interstitial fibrosis. It also showed foci of the intra-alveolar fibrous plugs consistent with foci of bronchiolitis obliterans organizing pneumonia (“BOOP”). The prosectors noted multiple foci of fibrotic hyalinized nodules with black anthrasic pigmentation involving approximately fifty percent (50%) of the lung parenchyma. The nodules were predominately micronodules with foci of macronodules, the largest of which measured 0.7 cm in greatest dimension. Multiple foci of squamous cell carcinoma were seen scattered throughout the lung parenchyma.

The clinicopathological summary stated that the 83 year old died with severe three blood vessels coronary artery disease. Micronodular CWP with focal macronodular CWP was seen. Metastatic squamous cell carcinoma in the lung was seen. It was added that due to the limitation of the autopsy, the primary site of the tumor could not be assessed.

Post-Mortem Reports

Dr. Everett F. Osterling

On August 27, 1999, Dr. Osterling, who is Board Certified in Anatomical and Clinical Pathology and Nuclear Medicine (see curriculum vitae at EX-2) submitted a medical report. (EX-1.) Dr. Osterling noted that he took thirty (30) photomicrographs of the autopsy slides. It is not clear from the report what other records were examined. He noted that the twenty-nine (29) autopsy slides were of excellent technical quality. He opined “it appears likely” the miner’s death primarily resulted from progressive systemic arteriosclerotic cardiac and cerebral vascular disease. In one photo, Dr. Osterling acknowledged the presence of a micronodule of CWP but noted it was pleural based. He noted the presence of pleural surfaces on opposite sides of tissue cross-sections in several cases. He added that a series of tissue samples were taken entirely from the pleura or the surface of the lung. He noted that it was unfortunate that this type of sampling was taken because the most severe changes in the lung occur within the pleura. Accordingly, he opined that the sampling in this case markedly accentuated the level of CWP and doubted that it reflected the true extent of the disease within the parenchyma. Dr. Osterling noted limited dust deposited within the pulmonary interstitium and the presence of passive congestion due to a failing left ventricle. He concluded that the primary cause of death was cardiovascular disease. A secondary “very” contributing cause was metastatic carcinoma. Dr. Osterling further noted it was *“difficult to assess the impact of his coal worker’s disease since the tissue slides do not appear to be representative of this gentleman’s functioning lung tissue.”* He noted the normal arterial blood gases and pulmonary function studies on 2-6-97 suggested the miner suffered little respiratory impairment based on his mine dust exposure. He added *he could not specifically answer employer’s questions related to the role of CWP* but expressed “doubt” it was in any way a significant contributing factor nor did it accelerate the miner’s death.

Dr. Osterling was deposed at length on May 24, 2000. (EX-7.) A list of documents reviewed by Dr. Osterling was submitted as an exhibit to the deposition transcript. He reiterated the findings in his report and once again noted *he could not determine the extent of the CWP due to the superficial sampling by the prosector at autopsy.* (TR 24). Dr. Osterling testified that the miner had normal vents and blood gases prior to death. He stated, “These [normal vents and blood gases] normally would not occur with the disease process of coal worker’s pneumoconiosis.” (TR 25). He added that if the disease was present there should be alteration in function. He noted the presence of CWP by autopsy and by x-ray. Dr. Osterling noted that even if squamous cell carcinoma were present, it would not be related to coal dust exposure. (TR 32). He opined that the miner did not have cor pulmonale but instead had left-sided heart disease. (TR 35-36). He noted that the cause of the miner’s death was failing left ventricle due to arteriosclerotic cardiovascular disease that had no causal connection to coal dust. (TR 37). Dr. Osterling opined that CWP did not cause, substantially contribute to, or hasten the miner’s death. (TR 38). He noted that the finding of severe coronary artery disease was not incidental and indicated his agreement with Dr. Griffin’s report. (TR 39).

On cross-examination, Dr. Osterling agreed that the autopsy slides were unrepresentative and that *he could not testify whether the miner had any pulmonary impairment or disability from the degree of disease identified because he had an unrepresentative sampling of tissue.* (TR 41). He stated that he believed the sampling reflected a higher level of disease than actually present, but, based on what he saw there was still not enough disease present to produce any significant respiratory alteration and would not have in any way contributed to the miner’s death. (TR 42). Dr. Osterling was reminded of his statement in his report that he could not comment on the impact of the CWP since the tissue slides did not appear to be representative of the miner’s lung and that he could not answer counsel’s questions regarding its role in the miner’s death. Nevertheless, Dr. Osterling was now able to proclaim with a reasonable degree of medical certainty that CWP did not contribute to the miner’s death. (TR 46-50).

Dr. Osterling admitted he did not conduct a gross review of the organs and was not present at the autopsy. (TR 50). He agreed that his opinion that the left ventricle was dilated was not supported by the prosector’s gross review that the cardiac chambers on both sides were unremarkable. (TR 51-55). Nevertheless, Dr. Osterling believed the dilation to be present based on his microscopic findings and other statements made by the prosector on gross. (TR 51-59).

Dr. Osterling noted that the measurement of the right ventricle by the prosector was done correctly. (TR 60). He noted that 0.5 cm was the upper limits of normal for the right ventricle. Although, Dr. Osterling was highly critical of the prosector’s description of the left ventricle on gross (i.e. cardiac chambers were unremarkable), he stated that he adopted the description of the right ventricle (i.e. as not being dilated) without question. (TR 61). He agreed that a diagnosis of cor pulmonale does not require dilation of the right ventricle. (TR 63-64). Dr. Osterling disagreed with Dr. Griffin’s assertion that cor pulmonale was not present because there was no dilation of the right

ventricle or right atrium. (TR 64). He disagreed with Dr. Griffin's opinion that the measurement of the right ventricle was taken incorrectly. (TR 64-65).

Contrary to the reports of Dr. Perper and the prosecutor, Dr. Osterling did not find microscopic evidence to support the existence of pulmonary hypertension. (TR 66). He admitted he did not notice in the miner's records an echocardiogram on 12-5-97 that was interpreted as showing "marked pulmonary hypertension" and left ventricle of normal size and thickness. (TR 67-68, 73). Dr. Osterling opined the miner did not have cor pulmonale because of the normal measurement of the valve and if changes in the left ventricle caused changes in the right it would be due primarily to cardiac disease and not pulmonary disease.

Contrary to the opinion of Dr. Griffin, Dr. Osterling did not diagnose idiopathic pulmonary fibrosis or diffuse interstitial lung disease. (TR 82-83). In support of his opinion, Dr. Osterling noted the miner's normal arterial blood gases ("ABG") from February 1997. He admitted he was unaware there were five (5) sets of arterial blood gases in the record and that four (4) sets produced values that were qualifying for disability under the Federal Regulations. (TR 87). He admitted the ABG values since 1979 were reflective of chronic hypoxemia. (TR 89). In addition, the miner had two (2) pulse oximetry studies (3-97 and 11-97) with reduced O₂ sats that Dr. Osterling was unaware of but admitted were reflective of pulmonary dysfunction. (TR 88). Dr. Osterling opined that the debilitating O₂ levels were primarily due to coronary artery disease and that the miner did not have significant CWP to cause these levels. (TR 90-91). He agreed that with an ejection fraction approaching forty percent (40%), six months prior to death, one would not anticipate hypoxemia to be present. (TR 91). After being advised that the February 1997 ABG he relied on in concluding there was no hypoxemia due to fibrosis was invalidated as physiologically impossible, Dr. Osterling admitted that this information would to a certain degree alter his assessment of the clinical records. (TR 92). Nevertheless, Dr. Osterling maintained that changes in tissue due to CWP were not that significant to explain the extent of hypoxemia present. (TR 97).

Dr. Osterling testified that only two (2) to three (3) tissue sections contained almost all lung parenchyma. (TR 105). He noted that nine (9) out of nine (9) macro photographs showed more pleura than should be present when doing a ninety (90) degree cut. (TR 110). He again noted that *there was enough sampling error to inhibit his ability to assess the severity of the disease.* (TR 113).

Dr. Osterling testified that he would defer to the pulmonologists and internists who concluded that in the miner's last two decades he had a true pulmonary dysfunction as opposed to a cardiac cause. (TR 122). Although it was not in his report, Dr. Osterling now concluded that the miner had a mild to moderate micronodular pneumoconiosis that he believed to be inflated due to sampling. (TR 123). Dr. Osterling admitted he did not see changes consistent with x-ray readings of 2/2 and 3/2 all six (6) zones. (TR 124). He could not explain what the radiologists were seeing. (TR 127). He

admitted that the x-ray changes were consistent with the degree of hypoxemia during those decades. (TR 128). He also admitted that right dilation due to a failing tricuspid valve could be an index of right ventricular failure. (TR 136).

Dr. John T. Schaaf

Dr. Schaaf, who is Board-Certified in Internal Medicine and Pulmonary Disease (see curriculum vitae at CX-4), submitted a medical report dated November 1, 1999. (CX-3). He reviewed the death certificate, Dr. Pisano's autopsy protocol and various medical reports and medical records. He did not review the autopsy slides. He concluded the miner had CWP based on radiographic and pathologic evidence and a comprehensive history of coal mine employment. He added that CWP was clearly a substantial contributing factor to the miner's death. Dr. Schaaf noted that physiologic impairment included pulmonary hypertension and hypoxemia due to CWP. In addition, the presence of CWP would also contribute to an increased susceptibility to acquiring pneumonia. He stated that the mechanism of death sounded much like a pulmonary death. The incidental findings of severe coronary artery disease and even his antecedent history of alleged congestive heart failure would not detract from the fact that CWP was a major contributing cause of death. He concluded that even if the terminal event were an episode of heart failure, the presence of hypoxemia and pulmonary hypertension would have both significantly contributed to making that condition worse.

The deposition of Dr. Schaaf was taken on May 1, 2000. (CX-10). Dr. Schaaf testified that he had evaluated the miner during his life and had diagnosed him as having pneumoconiosis that was later confirmed by autopsy. (TR 13). He opined the miner was not in congestive heart failure and had no significant smoking history to account for his breathlessness. (TR 14). Dr. Schaaf indicated that blood gas values obtained in 1997 were physiologically impossible as they were too high and not possible gases on room air. (TR 15). He agreed with Dr. Tuteur that these values should be disregarded as invalid. (TR 18). He added that the remaining ABG studies in the record showed significant to severe hypoxemia. (TR 18). Dr. Schaaf noted that the miner's coronary artery disease could not explain twenty (20) plus years of hypoxemia. (TR 23). The miner's valvular problems would not have caused hypoxemia because the heart valve has nothing to do with oxygen transfer. (TR 27). He concluded there was enough CWP present to cause elevated pulmonary pressure (TR 43) and that there was sufficient evidence pathologically and clinically of sufficient CWP to cause cor pulmonale. (TR 46). Dr. Schaaf admitted on cross-examination that congestive heart failure could be caused by severe coronary artery disease. (TR 81).

Dr. Joshua A. Perper

The medical report of Dr. Perper is dated November 5, 1999. (CX-1). He is Board-Certified in Anatomical and Forensic Pathology. He was the coroner of Allegheny County from 1982 to 1994. He is now the Chief Medical Examiner of Broward County, Fort Lauderdale. He has authored or co-authored nine (9) books and over one hundred (100) articles. (see curriculum vitae at CX-2). He prepared a thirty (30)-page report that included various supporting exhibits and articles on pneumoconiosis. He reviewed a list of fourteen (14) documents including the autopsy report, autopsy slides, medical records, and various medical reports. He summarized his review in detail. Dr. Perper noted a varied smoking history but in general the miner had a smoking history of three (3) to four (4) cigarettes per day for three (3) to twenty (20) years stopping thirty (30) to forty (40) years before his death. He detailed his review of the autopsy slides. He concluded there were three (3) joint causes of death; (1) hypertensive and arteriosclerotic coronary artery disease, (2) coal worker's pneumoconiosis with associated emphysema and chronic lung disease with cor pulmonale, and (3) metastatic lung cancer. Dr. Perper concluded that the moderate to severe pneumoconiosis was a substantial contributing factor in the miner's death through hypoxemia, both directly and indirectly in causing, precipitating or aggravating an arrhythmia in an individual with ischemic coronary artery disease and cor pulmonale. Moreover, based on a reasonable degree of medical certainty, Dr. Perper concluded that (1) the miner, with thirty-seven (37) years of coal mine employment, had sufficient evidence of complicated coal worker's pneumoconiosis on background and simple CWP with associated emphysema, chronic obstructive lung disease and cor pulmonale, (2) CWP was the result of thirty-seven (37) years of occupational exposure to mixed coal dust containing silica, (3) the miner's moderate to severe CWP and associated chronic obstructive lung disease and pulmonary cancer was a substantial contributor to the cause of death through contributing hypoxemia. Dr. Perper also opined that it was unlikely smoking played a significant role in the development of emphysema since the miner was a light smoker and quit many years before his death.

The deposition of Dr. Perper was taken on March 22, 2000. (CX-7). He reiterated his opinions and findings in his report. He noted the lung tissue sampling was done in a manner that reflected sufficient quantity of lung tissue, sufficient quality of lung tissue, correct procedure in sampling the lung tissue, and there was a representative sampling of lung tissue. (TR 18). Moreover he noted the tissue slides were consistent with the x-ray interpretations of record. (TR 20). He noted that there was sufficient sampling of organ tissues to assess the severity and nature of the disease process. (TR 12). Dr. Perper stated that Dr. Osterling's argument that the sections were not representative of the lung, was unreasonable and not logical. (TR 14). Dr. Perper added that the sections clearly represented both the pleura and a significant depth below. (TR 15).

Dr. Perper opined that cor pulmonale was present based on an increased heart weight of 480 grams and an increased thickness of the right ventricle (0.5 cm is slightly hypertrophic). The left ventricle was normal. (TR 23-24). Dr. Perper noted that cor pulmonale was substantially related to CWP and emphysema. (TR 29). He agreed with Dr. Pisano and was absolutely certain as to the presence of squamous cell carcinoma. (TR 25). He noted that squamous cell carcinoma may be

related to exposure to coal and silica but could not say within a reasonable degree of medical certainty. (TR 28).

Dr. Perper testified the records showed an early onset of a debilitating condition. Tests showed the presence of hypoxemia as early as 1979. He noted that over time the hypoxemia worsened to the extent the miner needed oxygen. (TR 30). Dr. Perper invalidated the arterial blood gas of 2-6-97 for three reasons: (1) it was impossible for the patient to have such high values, (2) if the values were normal, why would the patient need oxygen, and (3) high values were most likely due to patient being on oxygen. (TR 31). He also agreed with the invalidation of the study by Drs. Tuteur and Pisano. (TR 31).

Dr. Perper disagreed with Dr. Tuteur and stated there was no evidence of left ventricular dysfunction. (TR 38). He disagreed with Dr. Tuteur's conclusion that the miner had simple mild CWP and noted that Drs. Pisano and Osterling do not describe the disease as mild. (TR 42). He noted that Dr. Tuteur's conclusion did not correlate with the radiographic findings, arterial blood gas studies, and physician assessments. (TR 43). Dr. Perper agreed with the opinion of Dr. Schaaf. (TR 47-48).

Dr. James J. Pisano

The deposition of Dr. Pisano, the autopsy prosector, was taken on December 2, 1999. (CX-5). He testified that a resident, Dr. Magid Shenouda, assisted him during the autopsy. (TR 9). Dr. Pisano prepared the autopsy report with input from the resident. (TR 13). He noted that the left ventricle measured 1.3 cm and was normal. The right ventricle measured 0.5 cm indicating right ventricle stress and right ventricular hypertension. (TR 17). He noted it was ridiculous of Dr. Osterling to state the predominate number of tissue samples was from lung pleura. (TR 25). He reviewed the slides and indicated that if he had seen predominately pleural tissue he would have gotten more sections. (TR 28). He noted that he did not assess the pleura area for pneumoconiosis only the parenchymal tissue. (TR 29). Dr. Pisano testified that to his knowledge, neither he nor his resident intentionally selected pleura tissue to over-represent or inflate the severity of the disease. (TR 30). He added that he could not state for certain the source of the squamous cell cancer that was identified in the lung at autopsy. (TR 36). He did note that there was a link between silica and carcinoma of the lung. (TR 38). He noted that the degree of cancer present in the lungs at death would not have been present two (2) years before. (TR 45). Dr. Pisano stated that pneumoconiosis caused the interstitial lung disease that caused the miner's hypoxemia. (TR 43-44). He explained the relationship between pulmonary hypertension, cor pulmonale and hypoxemia. (TR 48). He noted that pneumoconiosis caused interstitial thickening which caused hypoxemia which caused pulmonary hypertension. (TR 49-50). Dr. Pisano concluded that pneumoconiosis was a significant contributing factor in that it was a major, substantial cause of hypoxemia which translated into ischemia of the heart. (TR 52).

On cross examination, Dr. Pisano stated with a reasonable degree of medical certainty that hypoxemia was the cause of death or ischemia of the heart secondary to coronary artery disease and pneumoconiosis of the lungs. (TR 76). He noted the miner had congestive heart failure which symptoms included difficulty breathing and fluid retention. (TR 87). He admitted the miner had left-sided heart failure. (TR 83). He described the miner's cancer as severe and by severe meant its metastasized but did not mean that it functionally interfered with the lungs much. (TR 86). He agreed there was no direct evidence that this cancer was related to coal mine dust exposure but again noted it was possibly related. (TR 87-88). Dr. Pisano agreed that the cancer may have been a contributor to miner's hypoxemia. (TR 90). He stated that the miner would have eventually died of squamous cell carcinoma without pneumoconiosis or coronary artery disease. (TR 92-93). He could not opine how long the miner would have lived if he did not have pneumoconiosis or coronary artery disease. (TR 93). Dr. Pisano testified the miner did not have complicated pneumoconiosis but simple pneumoconiosis based on the size of the micronodules present. (TR 104-105). Dr. Pisano opined the miner was on oxygen when arterial blood gases were performed in February 1997 because they were above the normal range. (TR 124).

Dr. Pisano submitted a supplemental report dated March 6, 2000. (CX-6). In his report he indicated that sixteen (16) autopsy slides or nineteen (19) sections contained lung tissue. Five (5) of the nineteen (19) sections primarily represented neoplasm. Fourteen (14) of the nineteen (19) sections were representative of the black lung process. Of the fourteen (14) sections that showed the black lung process, two (2) showed pleural nodules and twelve (12) showed almost all parenchymal tissue. Of the twelve (12) sections, seven (7) showed an edge with pleural surface and five (5) sections were completely without pleura. He concluded there were plenty of sections of lung parenchyma included among the slides to draw valid conclusions regarding the severity of the black lung disease in the decedent.

The second deposition of Dr. Pisano was taken on May 31, 2000. (CX-11). Dr. Pisano conducted a fourth (4) review of the autopsy slides. He noted that most of the slides showed 95% parenchyma. (TR 6). Dr. Pisano was given photographs of slides taken by Dr. Osterling, but they were unmarked and not numbered thereby making any comment by Dr. Pisano difficult. (TR 10-15).

Dr. Peter G. Tuteur

The medical report of Dr. Tuteur is dated January 31, 2000. (EX-3). Dr. Tuteur is Board-Certified in Internal Medicine and Pulmonary Disease. (see curriculum vitae at EX-4). He conducted an extensive medical record review including a review of the autopsy report, and medical reports of Drs. Osterling, Perper, and Pisano. Dr. Tuteur noted that the autopsy confirmed the presence of mild, simple coal worker's pneumoconiosis. The immediate cause of death was acute, superimposed on chronic congestive heart failure, with bilateral pleural effusions and hypoxemia. He added that the relevant studies of March 1997 and November 1997 demonstrated substantial interval worsening of

gas exchange. He invalidated the February 6, 1997 arterial blood gas as being physiologically impossible. Dr. Tuteur stated that the autopsy confirmed the diagnosis of histologically significant CWP and noted, "yet most reviewers consider this process mild and simple." Based on a reasonable degree of medical certainty, Dr. Tuteur opined that although there was evidence of a mild interstitial process neither the inhalation of coal dust nor the development of CWP played any role in contributing to or hastening death. He noted that the miner experienced exercise intolerance and breathlessness but that these were highly nonspecific symptoms consistent with any primary pulmonary, cardiac or neuromuscular disease. He added that the vascular disease, complications of diabetes mellitus, and carcinoma of the prostate were the major causes of the miner's breathlessness. He stated that the changes in pulmonary function were most compatible with worsening cardiac function. He commented on the articles cited by Dr. Perper in his report. Dr. Tuteur stated that although the miner had pathologically identifiable simple CWP, it was mild, focal and of insufficient severity and profusion to produce clinical symptoms, physical examination abnormalities, physical impairment or radiographic changes. He added that the significant problems adversely affecting the miner were totally unrelated to inhalation of coal mine dust or the development of CWP. These significant problems included: severe, advanced coronary artery disease resulting in ischemic cardiomyopathy with poor left ventricle function, congestive heart failure, pulmonary hypertension, tricuspid insufficiency complicated by diabetes mellitus with neuropathy, and angiopathy resulting in cerebral vascular disease. He added that carcinoma of the prostate diagnosed in 1995 was metastatic and was also a contributing and hastening factor of the miner's demise.

Dr. Steven P. Griffin

The medical report of Dr. Griffin is dated April 3, 2000. (EX-5). Dr. Griffin is Board-Certified in Anatomical and Clinical Pathology. (see curriculum vitae at EX-6). Dr. Griffin reviewed the medical records, autopsy report and slides. He disagreed with the diagnosis of cor pulmonale. He noted that the recorded right ventricle thickness of 0.5 cm was normal. He noted that the method of measurement, taken 2 cm below the pulmonary valve, was not normal. He added that the echocardiogram of 12-97 revealed a normal right ventricle. He stated that only five percent (5%) of the pulmonary parenchyma was occupied by CWP macules and micronodules. He added that the largest nodular masses were not squamous cell carcinoma but were due to metastatic adenocarcinoma with prostate origin. Dr. Griffin stated that the miner's shortness of breath and worsening leg edema was consistent with worsening congestive heart failure. He agreed with Dr. Osterling's assessment overall. Based on his review, Dr. Griffin concluded the miner had a relatively stable chronic pulmonary fibrosis with superimposed acute heart failure, metastatic carcinoma and pneumonia. He noted that the miner lived well beyond his expected life span and stated, "nothing in his health picture has accelerated his demise." He disagreed with Dr. Schaaf that the severe coronary artery disease was an incidental finding but was a primary finding and that congestive heart failure was diagnosed during the miner's lifetime.

Dr. Griffin concluded that the miner did have CWP. He noted the tissue showed “parenchymal collections of black pigment with interspersed birefringent crystals and fibrosis tissue forming coal worker’s macules and coal worker’s micronodules.” He noted the presence of scar emphysema comprising less than 1% of total emphysema which was primarily moderate in degree and mostly centrilobular. He opined his findings were consistent with mild, simple coal worker’s pneumoconiosis, micronodular. He added that CWP did not contribute to the cause of death of the miner. He stated that the CWP was simple, and occupied less than 5% of the pulmonary tissue and as such could not have contributed to his cause of death.

Dr. Griffin also concluded that the mechanism of death was congestive heart failure caused by severe coronary artery disease. The coronary artery disease was not related to or caused by CWP. He added that the metastatic carcinoma, which was not caused by CWP, was a significant factor and contributed to the miner’s death. Dr. Griffin opined that interstitial fibrosis with focal honeycombing was present but was not related to pigmented changes of CWP. This was the most likely cause of the respiratory function abnormality described by Dr. Strother. He also added that the underlying lung showed mild to moderate centrilobular emphysema not associated with black pigment and was not expected to be symptomatic due to mild to moderate degree. He added that the normal spirometry in 1997 was consistent with this assessment.

The deposition of Dr. Griffin was taken on July 27, 2000. (EX-8). Dr. Griffin reiterated the conclusions stated in his report. He stated CWP played no role in the process of death because it occupied so little of the pulmonary tissue. (July 27, 2000 deposition transcript page 39, 43). Moreover, he thought the miner’s interstitial pulmonary fibrosis with focal honeycombing was not related to the pigmentary changes of CWP and was probably the cause of the respiratory function abnormality described by Dr. Strothers. (July 27, 2000 deposition transcript page 53-54).

On cross-examination, Dr. Griffin admitted the records reflect the miner had some degree of pulmonary disability during his life that suddenly got worse the last two (2) years. He noted the miner’s x-ray findings were stable. (July 27, 2000 deposition transcript page 59-60). Although it was not noted in his report, Dr. Griffin agreed that the ABG study of February 6, 1997 was invalid. He stated that he referenced the study in his report because other pathologists did as well. (July 27, 2000 deposition transcript page 65). It was pointed out that Dr. Griffin, in his report, was referencing the “normal” February 6, 1997 ABG study to mark the sudden decrease in the miner’s condition in late 1997. (July 27, 2000 deposition transcript page 66). Dr. Griffin admitted that he called the values “normal” because Dr. Ignacio had called them normal. (July 27, 2000 deposition transcript page 74). In his report, after noting the “normal” arterial blood gases, Dr. Griffin stated, “this is not a pattern of severe coal worker’s pneumoconiosis which is relentless and progressive without episodic periods of normal pulmonary function studies and normal oxygenation.” On cross-examination, Dr. Griffin could not explain what “normal” values he was referring to. (July 27, 2000 deposition transcript page 77-78). Dr. Griffin agreed the arterial blood gas values reflected a mild degree of hypoxemia from the late 1970s. (July 27,

2000 deposition transcript page 92). But then he noted he would leave the degree of hypoxemia to pulmonologists to assess. (July 27, 2000 deposition transcript page 93).

Dr. Griffin opined the interstitial process was probably the cause the miner's pulmonary disability by and large, if not totally. (July 27, 2000 deposition transcript page 96). He admitted that no other pathologist of record supported his diagnosis of interstitial fibrosis with focal honeycombing. (July 27, 2000 deposition transcript page 101). He added he could not establish what percentage of the lung was involved with the interstitial fibrosis disease process because he was not present when the tissue was taken from the lung and did not know how much of the area was sampled. (July 27, 2000 deposition transcript page 103-104). Dr. Griffin maintained that he did not diagnose "idiopathic" pulmonary fibrosis in his report but diagnosed "interstitial fibrosis unrelated to dust exposure." (July 27, 2000 deposition transcript page 139). He added he was not willing to go any further in assessing the possible cause of the fibrosis. (July 27, 2000 deposition transcript page 140-141).

Dr. Griffin stated that the miner's pulmonary function studies showed a waxing and waning pattern. He based his opinion on abnormal spirometry and a normal pulmonary function study in 1997. (July 27, 2000 deposition transcript page 125). He noted an overall pattern of wellness that was not consistent with severe pneumoconiosis. (July 27, 2000 deposition transcript page 127). He agreed he did not evaluate pulmonologists opinions regarding the validity of the various vent studies in forming his opinion. (July 27, 2000 deposition transcript page 130). He agreed he would defer to a pulmonologist's opinion regarding the validity of the pulmonary function studies. (July 27, 2000 deposition transcript page 130). He could not opine whether interstitial pulmonary fibrosis could cause a waxing and waning of symptoms.

The deposition of Dr. Griffin was continued to September 13, 2000. At that time Dr. Griffin agreed that invalid vent studies should not be used to critique other physician opinions regarding the chronic nature of hypoxemia in this case. (September 13, 2000 deposition transcript page 20). He agreed he was able to classify the severity and degree of pneumoconiosis based on a review of the autopsy slides. (September 13, 2000 deposition transcript page 90). He did not recall any slides being all pleural. (September 13, 2000 deposition transcript page 96). He admitted he did not know what was causing the miner's hypoxemia in 1979 noting it could have been hypertension but doubted it. (September 13, 2000 deposition transcript page 143). He also noted that the chronic hypoxemia was of pulmonary origin. (September 13, 2000 deposition transcript page 149).

The continued deposition of Dr. Griffin was taken on November 9, 2000. He testified that his findings were consistent with the x-ray interpretations in the record in that his findings indicate the presence of simple pneumoconiosis. (November 9, 2000 deposition transcript page 16). He admitted there is some literature that causally links an increase in interstitial fibrosis with focal honeycombing to occupational exposure to coal dust. (November 9, 2000 deposition transcript page 25). He did not believe that pneumoconiosis could cause interstitial fibrosis. (November 9, 2000 deposition transcript

page 34). There was considerable discussion over the meaning of the phrase “idiopathic pulmonary fibrosis.” (November 9, 2000 deposition transcript page 65-78).

Dr. Waheeb Rizkalla

The medical report of Dr. Rizkalla is dated May 1, 2000. (CX-8). Dr. Rizkalla is Board-Certified in Anatomical and Clinical Pathology. (see curriculum vitae at CX-9). He reviewed the miner’s medical records and the autopsy protocol. It is not clear whether he reviewed the autopsy slides. He concluded the miner had pneumoconiosis and that the pneumoconiosis substantially contributed to his death by adding stress to his heart and by inducing hypoxemia and increasing the workload to his cor pulmonale and dilated right atrium thereby adding more strain to the severity of the atherosclerotic coronary artery disease. He added the area of malignancy of the lung was small and was considered an incidental finding and was not a substantial factor in the mechanism of death.

Conclusions of Law

Length of Coal Mine Employment

The parties have stipulated and I find that the miner was a coal miner within the meaning of the Act for at least 38 years. (TR 10).

Date of Filing

The parties have stipulated and I find that the claimant filed her claim for benefits under the Act on March 10, 1999. (DX-1; TR 9).

Responsible Operator

The parties have stipulated and I find that Keystone Coal Mining Company is the responsible operator and will provide payment of any benefits awarded to the claimant. (TR 10).

Dependents

I find that the claimant has no dependents for purposes of augmentation of benefits under the Act.

Date of Death

The parties have stipulated and I find that the miner died on July 31, 1998. (TR 9).

Presence of Pneumoconiosis

The parties have stipulated and I find the miner had pneumoconiosis arising out of his coal mine employment. (TR 10).

Applicable Regulations

Claimant's claim for benefits was filed on March 10, 1999 and is governed by the Part 718 Regulations. However, on January 19, 2001, substantial changes to Parts 725 and 718 of the Federal Regulations became effective. Based upon my review of the new Regulations there are two sections that specifically deal with the question of whether these new Regulations are applicable to cases that are currently pending at the time of the enactment.

Pursuant to § 725.2(c) the revisions of this part [Part 725] shall also apply to the adjudication of claims that were pending on January 19, 2001, except for the following sections: § 725.309, 725.310, etc.. (see the C.F.R. for the complete list of exempted sections). Accordingly, with the exception of those sections listed as an exemption, the revisions to Part 725 will apply to the facts of this decision.

Pursuant to § 718.101(b) the standards for the administration of clinical tests and examinations contained in subpart B "shall apply to all evidence developed by any party after January 19, 2001 in connection with a claim governed by this part [718]..." (emphasis added). Accordingly, since the evidence in the instant matter was developed prior to January 19, 2001, the newly enacted § 718, subpart B does not apply.

However, on February 9, 2001, the United States District Court for the District of Columbia issued a Preliminary Injunction in *National Mining Association v. Chao*, No. 1:00CV03086 (EGS) staying the implementation of many of the regulatory provisions issued by the Department of Labor in 20 C.F.R. Parts 718 and 725 on January 19, 2001. With respect to claims pending before the Office of Administrative Law Judges, the Court held, in pertinent part:

All claims for black lung benefits pending before the Department's Office of Administrative Law Judges at the time of this order...shall be stayed for the duration of the briefing, hearing, and decision schedule set by the Court, except where the adjudicator, after briefing by the parties to the pending claim, determines that the regulations at issue in the instant lawsuit will not affect the outcome of the case.

Accordingly, on February 15, 2001, I issued an Order pursuant to the Preliminary Injunction Order allowing the parties to submit briefs within ten days of receipt of the Order, addressing "with specificity whether the new regulations at 20 C.F.R. §§ 718.104(d), 718.201(a)(2), 718.201(c), 718.204(a), 718.205(c), or 718.205(d) will affect the outcome of [this] claim." Moreover, the Order

stated that “failure of a party to submit a brief is to be construed as a position that the amended regulatory provisions will not affect the outcome of the claim.”

The claimant filed her response on February 26, 2001. She indicated that it was unfair to be asked by the Court to speculate how the new regulations would affect the current proceeding when “[N]othing has been determined as to how the new regulations will be interpreted or applied by the Administrative Law Judge’s office, the Benefits Review Board, or the Third Circuit Court of Appeals.” Accordingly, the claimant requested a stay of the case until the Preliminary Injunction is made permanent or until the new rules take affect.

The Director, OWCP, a party-in-interest, filed a response on February 27, 2001. It was the position of the Director that none of the said amended provisions would affect the outcome of the instant matter.

The employer filed a response on April 19, 2001 and noted that “the Regulations at issue *may* affect the outcome of the current litigation.” (emphasis added). For this reason, the employer requested that this case be stayed until the issues being litigated before the District Court are resolved.

I disagree with the positions of the claimant and employer regarding their request for a stay and agree with the Director that the amended regulations will not affect the outcome in this case.³ Specifically, § 718.104(d) will not affect the outcome of this case because there is no report from the miner’s treating physician that was developed after January 19, 2001. 20 C.F.R. § 718.101(b). The revised, broadened legal definition of pneumoconiosis contained within Section 718.201(a)(2) will not affect this claim since there are no physicians of record who opine that coal mine dust exposure does not cause obstructive lung disease. Additionally, § 718.201(c) will not affect the outcome of this case because the existence of pneumoconiosis is not at issue. Section 718.204(c) is not relevant to this case because this is a claim for survivor’s benefits and therefore, total disability and disability causation are not at issue. Section 718.205(c) will not affect the outcome of this case because the provision is consistent with the existing case law in the Third Circuit. *See Lukosevicz v. Director, OWCP*, 888 F.2d 1001, 1006 (3d Cir. 1989) (holding that pneumoconiosis is a substantially contributing cause of a miner’s death whenever it hastens his death, even if a disease unrelated to pneumoconiosis also played a role.). Finally, §718.205(d) is virtually the same as the prior version with an update to the cross references. This will not affect the outcome of the claim.

³ In the Order, parties were to advise whether the amended Regulations “*will*” affect the outcome of this case. The employer’s response that application of said sections *may* affect the litigation does not meet the standard set forth in the Order.

Since I find that none of the amended regulatory provisions will affect the outcome of the claim, I will apply the remainder of the newly revised version of Part 718 (i.e. subparts A, C, and D) that took effect on January 19, 2001 to the facts of the instant matter.

Death Due to Pneumoconiosis

As noted earlier, on January 19, 2001, substantial revisions to Sections 718 and 725 of the Regulations became effective. Section 718.205 “Death Due to Pneumoconiosis” is essentially the same except for the addition of part 718.205(c)(5) which states:

Pneumoconiosis is a “substantially contributing cause” of a miner’s death if it hastens the miner’s death.

The comments to this section state, “§ 718.205(c)(5) simply codifies the Department’s longstanding interpretation of the legal standard for proving a miner’s pneumoconiosis was a ‘substantially contributing cause’ of his or her death under the BLBA and Part 718 regulations.....Section 718.205(c)(5) therefore represents a clarifying regulation which the Department may validly implement with retroactive effect for claims pending on the date the regulation becomes effective.” (emphasis added) (page 79950, Federal Register, Vol. 65, No. 245, December 20, 2000).

As noted in the comments, the change in the applicable section merely reflects the current state of the case law on this issue. Specifically, the Third Circuit Court of Appeals, which is controlling in this case, has adopted the “hastened death” standard in *Lukosevicz v. Director, OWCP*, 888 F.2d 1001, 1006 (3d Cir. 1989). Regardless of whether the parties agree or disagree as to the applicability of the new regulations, the same standard (“hastened death”) will be used in the instant decision in compliance with the rulings of the Third Circuit and new § 718.205.

The death certificate (DX-3), certified by Dr. Brent Ednie, indicated that the cause of the miner’s death was pneumonia and pulmonary hypertension. However, a death certificate, in and of itself, is an unreliable report of the miner’s condition and it is error for a judge to accept conclusions contained in such a certificate where the record provides no indication that the individual signing the death certificate possessed any relevant qualifications or personal knowledge of the miner from which to assess the cause of death. *Smith v. Camco Mining, Inc.*, 13 B.L.R. 1-17 (1989). Because the record contains no information that Dr. Ednie possessed any relevant qualifications or personal knowledge of the miner’s condition, I accord less weight to the death certificate.

There are seven physicians who have rendered opinions in this matter. Drs. Pisano, Schaaf, Perper, and Rizkalla opined the miner's pneumoconiosis was a substantial contributing factor in the miner's death. Drs. Osterling, Tuteur, and Griffin opined the miner's pneumoconiosis played no role in the miner's death.

I accord greater weight to the highly qualified opinions of Drs. Pisano, Perper, and Rizkalla. I find the medical opinion of Dr. Pisano, the prosector in this case, to be well-reasoned and supported by the objective diagnostic testing in the record as well as the gross and microscopic findings during the autopsy. Dr. Pisano was the only physician of record to conduct a gross examination of the relevant organs and tissue. Dr. Pisano testified at his deposition, with a reasonable degree of medical certainty, that hypoxemia was the cause of death or ischemia of the heart secondary to coronary artery disease and pneumoconiosis of the lungs. He explained the relationship between pulmonary hypertension, cor pulmonale, hypoxemia, and pneumoconiosis.

Likewise, I find Dr. Perper's medical report is well-reasoned, well-documented, and is substantially supported by the gross and microscopic findings detailed in the autopsy report. *Fields v. Island Creek Coal Co.*, 10 B.L.R. 1-19 (1987). At his deposition he explained the mechanics of death stating that the miner's moderate to severe pneumoconiosis contributed to the miner's death through hypoxemia both directly and indirectly causing, precipitating, or aggravating an arrhythmia in an individual with ischemic coronary artery disease and cor pulmonale. Dr. Perper's conclusion is supported by the objective diagnostic testing of record that showed hypoxemia as early as 1979, the gross findings during the autopsy that described black firm nodules occupying 50% of the pulmonary parenchyma, and the subjective complaints of the miner during his lifetime of longstanding shortness of breath. His findings substantially corroborate the opinion of the performing prosector, Dr. Pisano.

Although it is not clear from his report whether he actually reviewed the autopsy slides, I accord greater weight to the opinion of Dr. Rizkalla, who is also a Board-Certified Pathologist. He explained that the miner's pneumoconiosis substantially contributed to the miner's death by adding stress to the heart and by inducing hypoxemia and increasing the workload to his cor pulmonale and dilated right atrium thereby adding more strain to the severity of the atherosclerotic coronary artery disease. His opinion substantially corroborates the opinions of Drs. Perper and Pisano as to the mechanism of death.

Overall, I accord less weight to the opinions of Drs. Schaaf and Tuteur, who are both Board-Certified in Internal Medicine and Pulmonary Disease, since they are less qualified than Drs. Pisano, Perper, Rizkalla, Osterling, and Griffin, who are Board-Certified Pathologists, in assessing the cause of death. *Burns v. Director, OWCP*, 7 B.L.R. 1-597 (1984). They did not personally review the autopsy slides and relied, at least in part, on assessments made by other pathologists of record in determining a cause of death. Moreover, I particularly find that Dr. Tuteur's conclusions are not well-reasoned and do not correlate with the radiographic findings, arterial blood gas studies, and physician

assessments in the record. However, of relevance to the issue, both Drs. Schaaf and Tuteur invalidated the arterial blood gas study of February 6, 1997 as being physiologically impossible.

Conversely, I accord less weight to the opinions of Drs. Osterling and Griffin. Although they are highly qualified, I find the opinions of Drs. Osterling and Griffin to be inconsistent and inadequately reasoned. *Mabe v. Bishop Coal Co.*, 9 B.L.R. 1-67 (1986). Moreover, I find their opinions are equivocal and should be given less weight. *Griffith v. Director, OWCP*, 49 F.3d 184 (6th Cir. 1995); *Justice v. Island Creek Coal Co.*, 11 B.L.R. 1-91 (1988); *Parsons v. Black Diamond Coal Co.*, 7 B.L.R. 1-236 (1984).

Dr. Osterling maintained in his report and several times during his deposition that “it was difficult to assess the impact of his coal worker’s disease since the tissue slides do not appear to be representative of this gentleman’s functioning lung tissue.” Moreover, he stated in his report that he could not specifically answer the employer’s questions regarding the role CWP played in the miner’s death but expressed “doubt” that it was in any way a significant contributing factor. Then, at his deposition, although he again noted that he could not determine the extent of the miner’s pneumoconiosis due to superficial sampling by the prosector, Dr. Osterling was suddenly able to proclaim with a reasonable degree of medical certainty that CWP did not contribute to the miner’s death. No other physician of record supported Dr. Osterling’s opinion that the tissue sections were not representative of the lung and disease process. In addition, his report was inadequately reasoned in that Dr. Osterling relied on an invalid arterial blood gas study (February 6, 1997) to support his conclusion that there was no hypoxemia due to fibrosis. He admitted when confronted with this information that the vent study was invalid that it “threw a monkey wrench” into his assessment of the clinical records. Moreover, he could not give a reasonable explanation for the miner’s chronic hypoxemia dating back to 1979. Based on his inaccurate assessment of the clinical records and his unsupported allegations of the unrepresentative nature of the slides, Dr. Osterling’s opinion, which I also find to be equivocal, is accorded less weight.

Likewise, Dr. Griffin’s opinion should be given less weight. He noted the miner did not have cor pulmonale because the right ventricle thickness was normal. Contrary to the opinion of Dr. Osterling and others, Dr. Griffin maintained the prosector incorrectly measured the ventricle. He opined that the pneumoconiosis occupied less than five percent (5%) of the pulmonary tissue. That was significantly lower than the fifty percent (50%) estimate given by the prosector. No other physician diagnosed interstitial fibrosis with focal honeycombing except for Dr. Griffin. Like Dr. Osterling he relied on an invalid arterial blood gas study in early 1997 to support his conclusion that there was a sudden decrease in the miner’s condition in late 1997. Moreover, Dr. Griffin used this invalid study to critique other physician opinions that disagreed with his assessment. He could not establish what percentage of the lung was involved with the interstitial process but concluded anyway that it was “probably” the cause of the miner’s respiratory function abnormality. He could not identify a cause of the fibrosis except to say that it was not due to coal dust exposure. He could not identify the cause of the miner’s hypoxemia in 1979. For these reasons, I find that Dr. Griffin’s opinion is not persuasive and should be accorded less weight.

I find that the claimant has met her burden of showing that the miner's pneumoconiosis hastened his death under the standards imposed by § 718.205(c) and in *Lukosevich v. Director, OWCP*, 888 F.2d 1001, 1006 (3d Cir. 1989). Therefore, his death was due to pneumoconiosis within the meaning of § 718.205.

Entitlement

As the claimant has met her burden, she is entitled to benefits under the Act.

Date of Onset

Pursuant to § 725.503(c), benefits shall commence being paid beginning with the month of the miner's death, i.e. July 1, 1998.

Attorney's Fees

No award of attorney's fees for services to the claimant is made herein since no application has been received. Thirty days are hereby allowed to claimant's counsel for the submission of such application. His attention is directed to 20 C.F.R. 725.365 and 725.366 of the regulations. A service sheet showing that service has been made upon all parties, including the claimant, must accompany the application. Parties have ten days following receipt of such application within which to file any objections. The Act prohibits the charging of a fee on the absence of an approved application.

ORDER

The claim of Elsie Kozele s/s of Joseph Kozele for black lung benefits under the Act is hereby **GRANTED**; and

It is hereby **ORDERED** that Keystone Coal Mining Company, the Responsible Operator, pay to the claimant, Elsie Kozele, all benefits to which she is entitled under the Act, commencing as of July 1, 1998. and,

It is further **ORDERED** that Keystone Coal Mining Company reimburse the Secretary of Labor for payments made under the Act to the claimant, if any, and deduct such amount, as appropriate, from the amount it is ordered to pay under the preceding paragraph.

A

MICHAEL P. LESNIAK
Administrative Law Judge

MPL:mr

NOTICE OF APPEAL RIGHTS. Pursuant to 20 C.F.R. Section 725.481, any party dissatisfied with this Decision and Order may appeal it to the Benefits Review Board within 30 days from the date this

Decision and Order was filed in the office of the District Director, by filing a notice of appeal with the *Benefits Review Board at P.O. Box 37601, Washington, DC 20013-7601*. A copy of a notice of appeal must also be served on Donald S. Shire, Esq. Associate Solicitor for Black Lung Benefits. His address is Frances Perkins Building, Room N-2117, 200 Constitution Avenue, NW, Washington, D.C. 20210.